In vivo and in vitro Deiodination of Silver Iodide

Silver iodide is the most water-insoluble salt known (0.03 mg/l) and shows a remarkable resistance to a chemical attack. As most of the silver salts, it is darkened by the UV-light in a photochemical reaction which releases iodine[1]. In spite of its chemical stability this compound is readily broken down in presence of organic tissues.

This experimental work has been performed to study the deiodination in vivo, as well as in vitro, of particulate ^{131}I-labelled silver iodide. The radioactive material was prepared as follows: 2 ml of 0.01 *M* $Na^{131}I$ were precipitated with 0.2 ml of 0.1 *M* $AgNO_3$. Immediately after the precipitation, 0.5 ml of 10% gelatin solution was added, and after heating for 5 min in a boiling water-bath it was centrifuged at 2000 rpm during 2 min. The colloidal particles which must be discarded, are brought in suspension by the gelatin. After washing the precipitate 3 times with distilled water and repeating the centrifugation under the same conditions, the sediment was resuspended in 0.5 ml of 10% gelatin. Finally, it was diluted to 20 ml with saline. The silver iodide prepared in this way has a particle size range from 0.4–1.5 μ.

Young albino mice (15–20 g) were injected into the tail vein with 0.2 ml of $Ag^{131}I$ suspension (0.23 mg AgI/ml and 10 μC ^{131}I/ml). Groups of 3 animals were sacrificed at different intervals. After evisceration, the radioactivity in liver, stomach and thyroid was counted with a well-type scintillometer. A similar experiment was carried out using silver iodide labelled with ^{110}Ag. This material was prepared using the same technique, but with radioactive silver nitrate ($^{110}AgNO_3$). Table I shows the results of both experiments, as the mean value of the radioactivity in 3 animals; 10 min, 30 min, 1 h, 3 h, 6 h and 24 h after the i.v. injection of the radiocompound.

An in vitro assay was performed to study the stability of ^{131}I-labelled silver iodide. The tested mouse tissues were homogenized with Ringer's solution to give a final tissue concentration of approximately 100 mg/ml (wet weight). 2 ml of homogenate were incubated during 3 h with 0.3 ml of Ag ^{131}I. This experiment was done in triplicate. In each series a blank containing the same amount of radioactive silver iodide, but with Ringer's solution instead of homogenate was incubated under the same conditions (3 h at 37 °C).

In order to test if a thermostable factor is responsible for the deiodination, another series of assays was carried out but heating the homogenate previously the incubation (10 min at 80 °C).

After the incubation was completed, 0.2 ml of 10% human serum albumin was added to each tube and immediately precipitated with 2.3 ml of 10% trichloracetic acid solution. The albumin is added to eliminate by flocculation any silver iodide that could be peptized during the incubation. After centrifugation, the radioactivity was counted on an aliquot of the supernatant. Table II shows the results of these experiments as the mean value of 3 determinations and as % of radioiodine released.

The in vivo results indicate that after 6 h approximately 83% of the radioiodine has been eliminated from the liver, but only 16% of the silver. The increasing radioactivity in stomach and thyroid while it decreases in liver, is an indication that the radioiodine is set free as soluble iodide. On the other hand, the decrease of radiosilver observed in liver after 24 h (Table I), could be explained by its excretion through the bile[2].

The in vitro results suggest that a thermostable factor is responsible for the silver iodide break-down. The differences observed with and without preheating, could be related more likely to a surface action difference (coagulation) than to a denaturation by heating.

These experimental findings are in agreement with the already studied in vitro deiodination of ^{131}I-labelled oleic acid and triolein. In this case, also was found a higher dehalogenase concentration in liver and blood[3]. Furthermore, it seems likely that a similar thermostable factor is responsible for the deiodination of both silver iodide and iodinated organic compounds[4,5].

Table I. Distribution of the radioactivity at different times after the i.v. injection of ^{110}AgI and $Ag^{131}I$ (as % of the injected dose)

	10 min	30 min	1 h	3 h	6 h	24 h
After i.v. injection of $Ag^{131}I$						
Liver	70	59	51	24	12	4.1
Stomach	4.2	7.3	8.0	17	6.1	0.2
Thyroid	0.9	1.2	2.1	6.3	6.5	5.8
After i.v. injection of ^{110}AgI						
Liver	53	56	54	53	44	21
Stomach	0.2	0.3	0.4	0.4	0.6	0.3
Thyroid	0.2	0.3	0.2	0.4	0.5	0.3

Table II. ^{131}I-iodide released after 3 h of incubation (as % of the ^{131}I as $Ag^{131}I$ incubated)

	Liver	Stomach	Muscle	Blood
Normal tissue	28	6.8	15	13
Preheated tissue	18	1.7	10	13

Résumé. La stabilité de l'iodure d'argent a été étudiée in vivo, par rapport à l'élimination de l'argent et du iode radioactif. Le foie semble responsable de la désiodation. Dans le foie, au bout de 6 h, 83% de l'iode est eliminé, mais seulement 16% de l'argent. Les expériences in vitro indiquent que le facteur responsable de la désiodation est thermostable et plus concentré dans le foie et dans le sang.

L. J. Anghileri[6]

Deutsches Krebsforschungszentrum, Institut für Nuklearmedizin, Heidelberg (Germany),
4 April 1968.

[1] K. H. Lieser, E. Lakatos and P. Hoffman, Ber. Bunsenges Phys. Chem. *70*, 176 (1966).
[2] L. J. Anghileri and H. Harbst, J. nucl. Biol. Med. *11*, 107 (1967).
[3] L. J. Anghileri, Nature *208*, 896 (1965).
[4] P. Raban, V. Gregora and M. Jirsa, Nature *204*, 885 (1964).
[5] G. M. de Escobar, F. E. Del Rey and P. L. Rodriguez, J. biol. Chem. *237*, 2041 (1962).
[6] Present address: The Johns Hopkins Medical Institutions, Department of Radiological Science, Baltimore (Maryland 21205, USA).